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An elderly woman with a warm, painful finger

QUESTION: A 91-year-old Russian-speaking woman arrived by ambulance to the emergency department because she was having severe pain in her right middle finger. She has had swelling of her finger for about a year. Her physician had referred her to a dermatologist 2 weeks earlier, who started her on a regimen of warm soaks and an antibiotic. Two days earlier, the physician had attempted incision and drainage, but no purulent material was obtained. Physical examination revealed the finger shown in figures 1 and 2. Palpation of the distal interphalangeal joint demonstrated firmness rather than fluctuance.

What is the differential diagnosis, and what would you do next?

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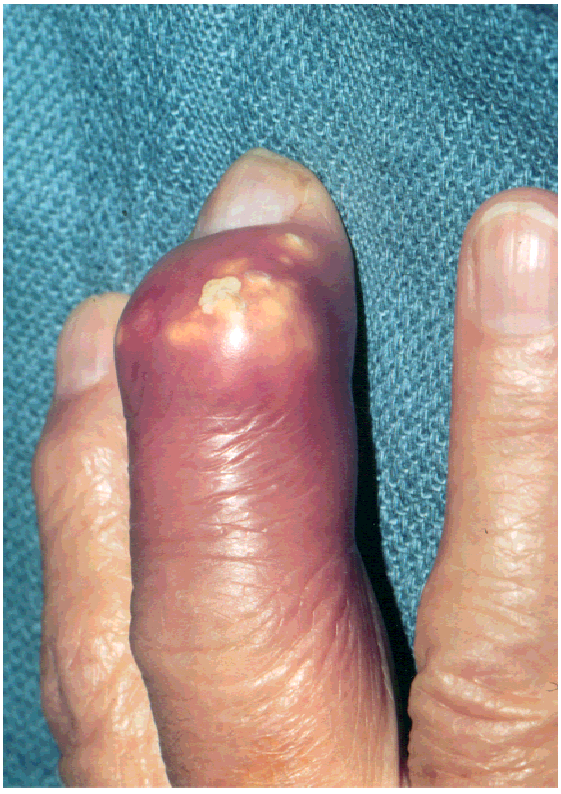

Figure 1

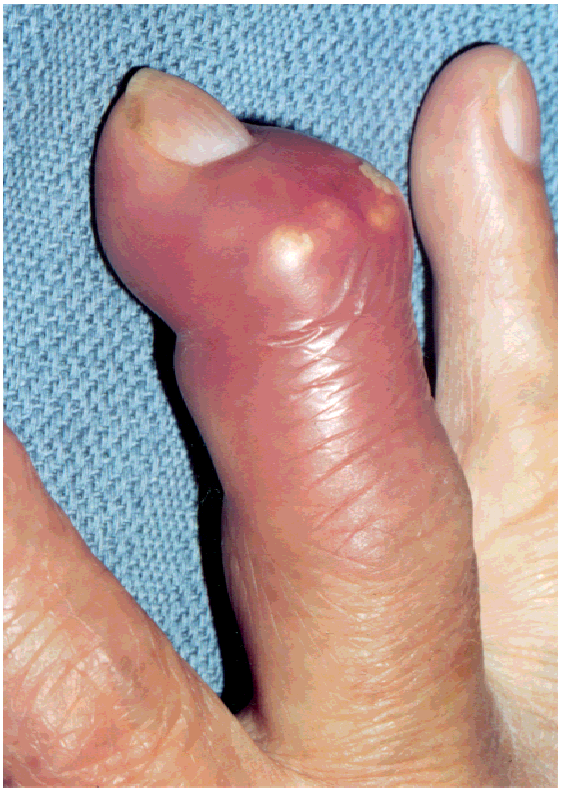

Figure 2

ANSWER: A radiograph of the finger is shown in figure 3.

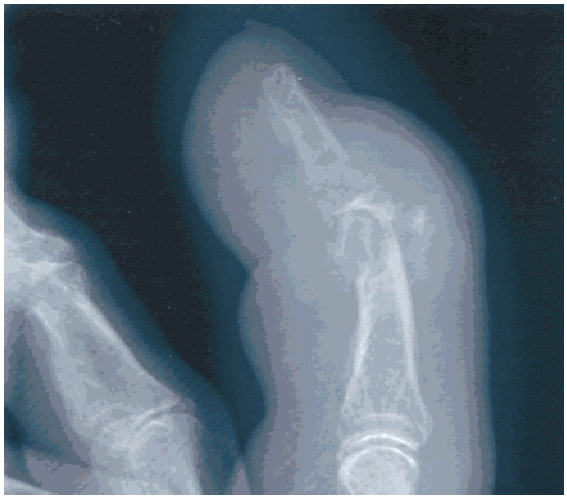

Figure 3

The findings of the radiograph and physical examination are consistent with acute gouty arthritis superimposed on tophaceous gout. The diagnosis was confirmed by an aspirated specimen from the finger that showed, both intracellularly and extracellularly, negatively birefringent, needlelike crystals. After consultation with a rheumatologist, a regimen of colchicine, 0.6 mg every 6 hours, was started. Her pain was markedly decreased after 24 hours. Her serum uric acid level was 0.64 mmol/L (10.7 mg/dL). She was referred for management of her hyperuricemia.

EPIDEMIOLOGY

Gout is classically a disease of adult men, with a peak incidence in the fifth decade of life. From 10% to 25% of patients have a family history of gout. The incidence of gout in women increases in the postmenopausal period when the uricosuric effect of estrogen is lost.[1]

The clinical characteristics of gout in the elderly differ appreciably from those in younger patients (see box).[2]

PATHOPHYSIOLOGY

Gout is manifest by acute attacks of crystal-induced arthritis and by ongoing deposits of monosodium urate, or tophi, in and about the joints and subcutaneous tissue.[3] Hyperuricemia leads to gout, although this complication occurs in only a few patients with elevated urate levels. Primary gout is the result of an inborn error of purine metabolism, leading to increased production of uric acid. Secondary gout results from either increased production in certain disease states or as the result of decreased excretion, either from disease state or drug therapy. Tophaceous gout is a consequence of the long-term inability to excrete urate as rapidly as it is produced.[4] Tophi development is more likely to occur the longer hyperuricemia is present and/or the higher the serum urate level becomes. The yellow-white patches visible in the finger in Figures 1 and 2 are the tophi. The violaceous skin color shown in the photographs is typical of the overlying skin seen in acute gouty arthritis.

DIFFERENTIAL DIAGNOSIS

Other causes of acutely swollen, painful joints include infections such as cellulitis, septic arthritis, osteomyelitis, or septic bursitis; pseudogout (calcium pyrophosphate crystals); calcific periarthritis; joint trauma; or other inflammatory arthritides. The presence of tophi, if recognized, is a hallmark of gout.

TREATMENT

In young healthy patients who have no contraindications, first-line therapy for acute gouty arthritis is usually with nonsteroidal anti-inflammatory drugs.[5] These drugs are rapidly effective in most patients in relieving pain and reducing inflammation, especially if they are taken soon after the onset of an attack. A second-line therapy for an acute attack of gout is colchicine. The use of nonsteroidal anti-inflammatory drugs is fraught with problems in older patients. Colchicine therapy may also present unacceptable risks in an older patient, especially when given intravenously. In addition to gastrointestinal intolerance, colchicine use may exacerbate renal failure or produce bone marrow suppression, hepatic necrosis, or seizures. Corticosteroids are sometimes used intra-articularly in patients with acute monoarticular gout.

Serum urate levels may be brought down using the xanthine oxidase inhibitor, allopurinol.[5] Such therapy is primarily indicated for patients with recurrent attacks of gout, urolithiasis, myeloproliferative disorders, or tophi. Urate-lowering therapy should not be initiated or discontinued during an acute attack because it can exacerbate the attack.[4,5]

Uricosuric drugs may be used in patients with chronic tophaceous gout.[1,4] Patients with massive tophaceous deposits and normal renal function may respond to a combination of allopurinol and a uricosuric agent. Surgical excision of tophi is occasionally indicated.[3]

Gout in elderly patients

- There is a more even sex distribution than in younger patients
- Gout in these patients is frequently associated with long-term diuretic use, excessive alcohol consumption, and renal insufficiency
- Gout in these patients primarily results from underexcretion, rather than overproduction, of uric acid
- Acute painful arthritis attacks occur less frequently, and tophus formation is more common in the elderly

References

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